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NON-ORGANIC CONVULSIVE DISORDERS OF CHILDHOOD—WITH SPECIAL REFERENCE TO IDIOPATHIC EPILEPSY*

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I

MEDICAL HISTORY affords no more interesting nor more tragic chapter than that concerning the convulsive disorders of children. This is particularly true of epilepsy, which, because of its chronicity and its intractability in the face of all efforts at treatment, has apparently puzzled and plagued mankind everywhere since the very dawn of the human race. Even in this enlightened day, most physicians, as well as laymen, rightly regard a severe generalized convulsion with alarm, and are filled with despair when the symptom recurs in chronic form. The basis for this attitude is to be found not only in the awareness that a convulsion may herald the onset of certain extremely serious diseases, but even more in the glaring deficiency of our knowledge regarding the causative mechanisms in those chronic convulsive states for which no structural pathology can be demonstrated.

A general survey of the convulsive disorders seen over a period of years in any large pediatric clinic of the temperate zone would probably show a distribution of cases on the basis of etiology and age not significantly different from that shown graphically in Figure 1. Here it may be observed that the relative frequency of certain etiologic factors varies greatly with age. Birth injuries and congenital defects of the brain account for a large majority of cases during the first month of life, acute infections playing a secondary rôle. During the next three months the order is reversed. Throughout the remainder of infancy, and in very early childhood, infantile or rachitic tetany assumes an important position. During the age period immediately above this, the acute infectious diseases again play the major rôle, with idiopathic epilepsy also accounting for a significant percentage of cases for the first time. Throughout the remainder of childhood—that is, beyond the sixth year—the latter disorder is by far the most frequent cause of convulsive seizures.

Granting that a small percentage of the patients here classified as idiopathic epileptics might be found by careful encephalographic or postmortem studies to have structural lesions in or about the brain, it is generally conceded that the great majority show no such changes. This, then, brings into numerical prominence the entire class of convulsive disorders for which no definite brain pathology has been demonstrated. The present paper deals primarily with this type of disorder. (See shaded portion of charts in Figure 1.) The chief representatives of the non-organic convulsive group are the following: infantile and other forms of tetany, hypoglycemic states, uremia, acute poisoning with certain convulsant drugs, acute infections not directly affecting the brain or meninges, certain conditions involving impairment of the circulation to the brain, and finally, idiopathic, cryptogenic or genuine epilepsy. While the exact physiologic mechanism of the convulsions is not as yet known for any of these conditions, recognition of the fact that an organic lesion of the brain is not essential for the occurrence of either *petit mal* or generalized seizures, and that physiological or chemical disturbances can in themselves be the cause of seizures, is undoubtedly a first step toward a solution of the problem involved.

In spite of the fact that the primary etiologic factor is different in each of the above-named conditions, the generalized convulsion itself appears outwardly to be the same in all cases. From this we might expect certain features of the underlying physiological mechanism to be the same in all instances, and such actually appears, from the limited data available, to be the case. These common features, as well as certain differences, will be referred to later. Since time does not permit a detailed discussion of the mechanism and treatment

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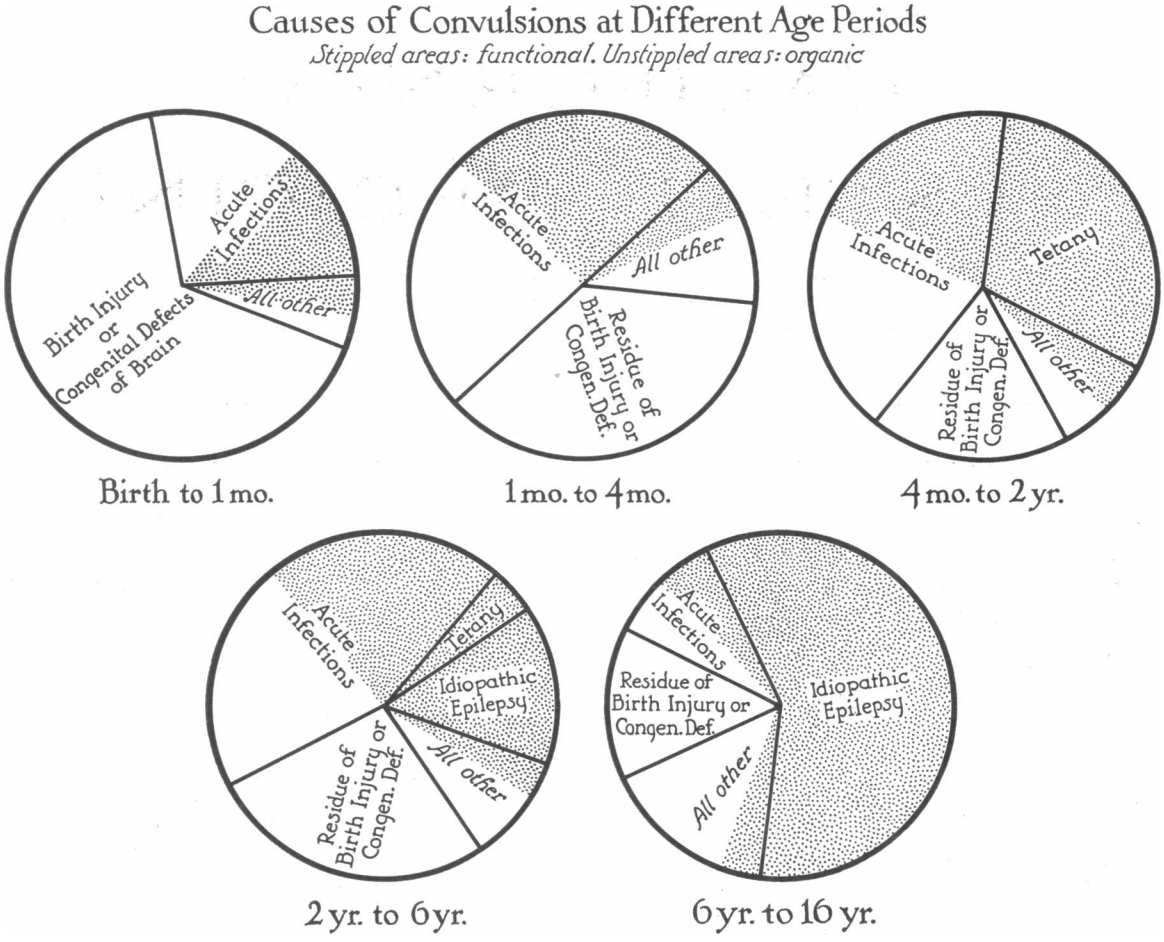

Fig. 1.—Schematic representation of the etiological distribution of patients admitted to the average pediatric clinic with the complaint of convulsive seizures.

of each entity under consideration, the results of recent studies on idiopathic epilepsy, the least well understood but most important representative of the group, will be presented.

In the way of definition, the admittedly unsatisfactory term "idiopathic epilepsy" will be employed to denote a particular type of convulsive disorder which is characterized by its chronicity and by the obscurity of its underlying etiology. The current fashion of using the term in a generic sense to group together all conditions in which convulsions occur is confusing and leaves the particular type of case under discussion inadequately defined. While it is true that the size of the group once classified under the diagnosis of idiopathic epilepsy has gradually been diminished with advances in our knowledge, the culling process has only increased our awareness of the fact that there still remains a large number of clinically similar cases, having in common an abnormal proneness to convulsive seizures for which no organic or physiological basis has as yet been demonstrated. In the following pages the two closely related aspects of the epilepsy problem, etiology and treatment, will be considered.

ETIOLOGY

The intrinsic nature of the subtle constitutional factor, which is responsible for the epileptic's characteristic proneness to seizures, constitutes the unsolved etiologic problem of this convulsive state. No matter from what angle the problem is approached, one is invariably led to the same ultimate conclusion, that the chronic spasmophilic tendency is due to an inherent abnormality in the mode of functioning of the brain cells. There may well be some underlying defect in the chemical structure of these cells to account for their erratic behavior; but thus far, histochemical technique has not been sufficiently refined to detect this. Some indirect evidence referred to below indicates that the characteristic deficiency is one relating to the semipermeability of the brain cell membrane.

Whatever the nature of the abnormality may be, it appears to be firmly fixed in the germ plasm, and shows an unmistakable tendency to pass from generation to generation by inheritance, as shown by the recent report of Lennox and Cobb.[1] The inheritance factor appears from the work of these authors to be vastly more important in cases of epilepsy beginning in childhood than in those having their onset after the age of twenty years. Davenport and Weeks[2] regard it as a Mendelian recessive due to "absence of a protoplasmic element."

CONTRIBUTING ETIOLOGIC FACTORS.—In addition to the underlying abnormality just referred to as being responsible for the increased "con-

TABLE 1.—*Average Values Per Hundred Grams of Various Foods Used in the Ketogenic Diet*

Food Material (Edible Portion, Fresh State)	Fat Gm.	Protein Gm.	Carbohydrate Gm.	Calories	Water Gm.	Excess in Ash	
						Acid cc. 0.1 N	Alkali cc. 0.1 N
Forty per cent cream	40	2	3	388	54	....	25
Butter	85	....	....	790	15	....	....
Special French dressing	62	....	2	585	35	90	....
Special mayonnaise dressing	67	3	1	640	28	77	....
All food oils	100	....	....	930	....	....	....
Bacon—medium lean	65	10	....	645	20	65	....
Pork chops or fresh ham—medium fat	25	16	....	300	54	95	....
Beef, mutton or turkey—medium fat	20	18	....	260	60	110	....
Chicken, fish, wild game, tongue, veal—lean	5	19	....	125	72	125	....
Shell fish	1	16	3	87	80	90	....
Cheese—American, Cream, Swiss	33	28	1	420	30	....	6
Cottage cheese	1	21	4	115	72	....	40
Milk "substitute"	20	4	1	204	74	....	50
Casein (Casec)	1	88	....	366	....	....	....
Gelatin (dry or D'Zerta)	....	84	....	340	14	....	....
Eggs, hens'	12	13	....	164	73	160	....
Egg white	....	12	....	49	86	52	....
Egg yolk	33	16	....	372	49	267	....
Brazil nuts	67	17	7	721	5	....	50
Butter nuts	61	28	4	698	4	....	50
English walnuts	64	18	12	718	3	....	65
Green olives	13	2	2	137	75	....	200
Avocado (alligator pear)	23	2	7	243	66	....	75
"Five per cent" vegetables and fruits: (Lettuce, cucumbers, spinach, asparagus, beet greens, celery, mushrooms, watercress, cabbage, radishes, grapefruit, rhubarb, tomatoes)	....	1	3	16	92	....	65
"Ten per cent" vegetables and fruits: (String beans, squash, turnips, beets, cauliflower, onions, carrots, pumpkin, lemons, oranges, strawberries, muskmelon, peaches, pineapple, blackberries, watermelon)	....	2	6	32	88	....	75
"Fifteen per cent" vegetables and fruits: (Green peas, parsnips, young lima beans, artichokes, raspberries, currants, apricots, pears, apples, blueberries, cherries)	....	2	15	69	80	....	65
Yeast powder (Mead's)	1	48	39	280	5	....	....

vulsive reactability," there are numerous factors which may contribute to the occurrence of seizures, presumably by accentuating the abnormal tendency already present. Chief among these are brain trauma, emotional disturbances, any impairment of the intracranial circulation, anoxemia, shifting of the acid-base equilibrium toward the alkaline side, lowering of the ratio of cholesterol to lecithin in the blood plasma and, finally, a positive water-balance with dilution of the body fluids.

Injury to the Brain.—Injury to the brain and its coverings is undoubtedly a potent factor in bringing out the latent tendency to seizures. Reflex or direct irritation, whether mechanical, toxic or infectious in origin, tends to lower the convulsive threshold. Emotional upsets frequently have a similar effect, particularly in some cases of the *petit mal* type. The possibility has been suggested by Cannon that strong emotion, having its neural basis in the diencephalon, might cause discharges of impulses from the latter to the motor cortex.

Disturbances of Intracranial Circulation.—That disturbances of intracranial circulation may play a prominent part in the causation of seizures has long been suggested by the fact that the skin and

TABLE 3.—*Correlation Between Changes in Plasma Lipids and Occurrence of Convulsions on Various Dietary Regimens*

Successive Regimens (H_2O ad lib.)	Total Fatty Acids	Lecithin	Cholesterol	Lecithin Cholesterol	Convulsions
Ordinary Mixed Diet	210 220 259	102 103 99	83 80 79	1.25 1.28 1.25	Several daily
After 11 days on F., 149; C., 9; P., 36 g. (acid ash)	270	125	113	1.10	None for seven days
Temporary high carbohydrate intake	340	141	95	1.49	Two hours before and one hour after sample
After 5 days on F., 149; C., 9; P., 36 g. (alkaline ash)	313	151	102	1.38	Sixteen during five days
After 6 days on F., 170; C., 0; P., 55 g. (ketogenic)	383	175	208	0.84	None for five days before and three days after sample
After 3 days on F., 110; C., 0; P., 200 g. (non-ketogenic)	460	225	133	1.69	One hour before and one hour after sample

mucous membranes of some epileptic subjects are known to become pale just prior to the onset of a convulsion. During brain operations under local anesthesia, Kennedy, Penfield, Foerster and others have repeatedly observed blanching of the exposed brain at the onset of generalized convulsions; which suggests that reflex spasm of the cerebral arterioles might be an important factor in some instances. The objection to this being regarded as the primary cause is its inconstancy and the lack of proof that it is not itself an initial effect of the seizure, rather than its cause. Another obstacle in the way of accepting the circulatory theory of etiology for epilepsy is the recent finding of Cobb, Gibbs, and Lennox [3] that the volume flow of blood through the brain in relationship to seizures is not abnormal, as determined by the use of a delicate thermo-electric blood-flow recorder. While severe asphyxia may cause convulsions, there is no outward evidence for the existence of this condition before the onset of a genuine epileptic seizure, and temporary mechanical pressure applied to the jugular veins is apparently no more likely to induce a convulsion in an epileptic than in a normal subject. While dysfunction of the *vegetative nervous system* might be expected to play an accessory part in the seizure mechanism of idiopathic epilepsy through interference with the circulation to the brain, the small group of patients described by Weiss and Baker [4] as having convulsive reactions or fainting attacks due to hyperactivity of the carotid sinus reflex cannot be regarded as genuine epileptics, because seizures can be induced in the latter but rarely, if ever, by pressure over the sinus.

Endocrine Function. — That some abnormality of the closely related endocrine functions might contribute to the onset of epileptic attacks is naturally suggested by the fact that hyperinsulinism (or its equivalent, the underaction of the diabetogenic hormone of the hypophysis) and hypoparathyroidism, are both known to cause convulsive seizures in the absence of any pathological lesion in the brain. As will be shown under another heading, seizures can be induced almost at will in epileptic subjects by administration of antidiuretic pituitary extract, if this is given under conditions favorable to the establishment of a strongly positive water-balance. Overdosage of epinephrin will also cause convulsions in normal animals. However, there is no convincing proof that any of the glands of internal secretion are characteristically abnormal in epilepsy.

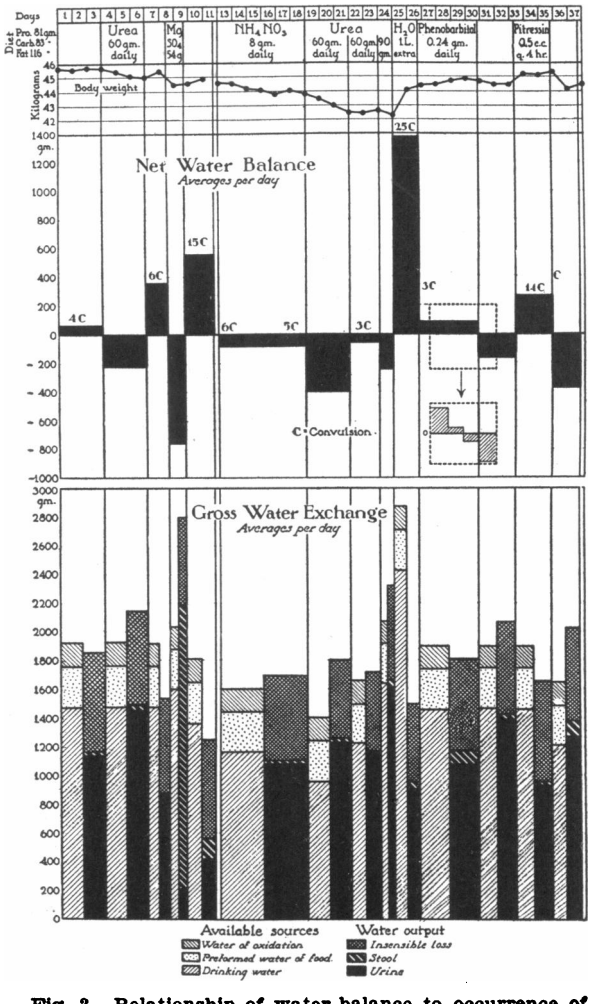

Fig. 2.—Relationship of water balance to occurrence of convulsions (C) under various conditions.

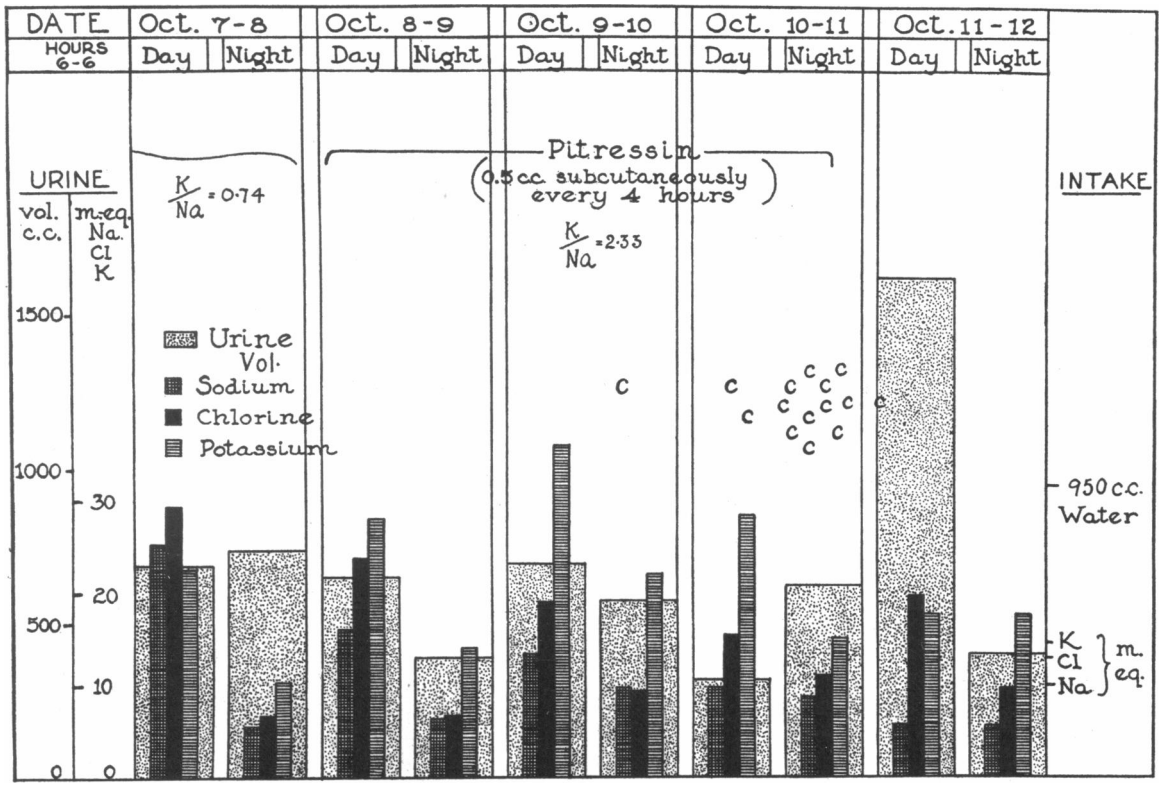

Fig. 3.—Water, Na, K and Cl balances in relationship to occurrence of convulsions (C) induced by sustained pituitary antidiuresis.

Anoxemia.—Anoxemia is thought to be the important abnormality responsible for the convulsions of cerebral anemia or asphyxia. Lennox [5] has found that the ease with which seizures can be induced in epileptic patients by voluntary hyperventilation of the lungs is greatly increased if the subject is placed in an atmosphere with reduced oxygen tension. Oxygen tensions above those of the ordinary atmosphere lessen the tendency for seizures to follow hyperventilation, but do not appear to inhibit spontaneous seizures. Up to the present time, however, no characteristic abnormality of the respiratory functions has been discovered in epilepsy. Nevertheless, any serious interference with the supply of oxygen to the brain cells tends to favor the occurrence or seizures, unless this effect is simultaneously offset by an increase in the hydrogen ion concentration of the system, as it may be when air is rebreathed.

Acid-Base Equilibrium. — That the acid-base equilibrium of the body fluids might bear some relationship to the occurrence of convulsive seizures was suggested by the well-known fact that alkalosis increases, while acidosis decreases nervous irritability. Certain workers in Copenhagen, and later several in America, presented evidence of an unstable acid-base equilibrium in epileptic patients, which was most noticeable just prior to the onset of seizures. More dependable proof than that afforded by these observations, however, is the well-recognized fact that procedures which cause alkalosis (such as the administration of alkaline salts, hyperventilation of the lungs and prolonged vomiting), tend to induce seizures; while those factors which cause acidosis (such as diarrhea, fasting, a ketogenic diet, ingestion of mineral acids or acid-forming salts and breathing an atmosphere high in CO_2) all favor the cessation of seizures. The convulsion itself is terminated by the development of the endogenous acidosis arising from retention of CO_2 and extra production of lactic acid from violent muscular contractions. Alkalosis not only increases nervous irritability, but may interfere with brain circulation by virtue of the fact that it causes contraction of the smaller arterioles.

Lipid Economy.—Recent studies on the lecithin, cholesterol and total fatty acids of the blood in relationship to seizures indicate that there may be some obscure abnormality in the lipid economy in epilepsy.[6] While no constant relationship has been found to exist between the absolute level of any lipid fraction and the occurrence of seizures, the lecithin-cholesterol ratio shows a striking tendency to be higher at, or near the time of convulsions than at other times. (See Table 3.) This tendency for seizures to be related to high lecithin and low cholesterol concentrations may find a rational interpretation in the known effects of these two physiologically antagonistic substances, which occur in unusually high concentration in brain cells. Cholesterol, when injected intravenously, produces profound narcosis and active diuresis. Lecithin exhibits almost opposite effects, for, when it is administered parenterally in conjunction with a narcotic, it shortens the period of

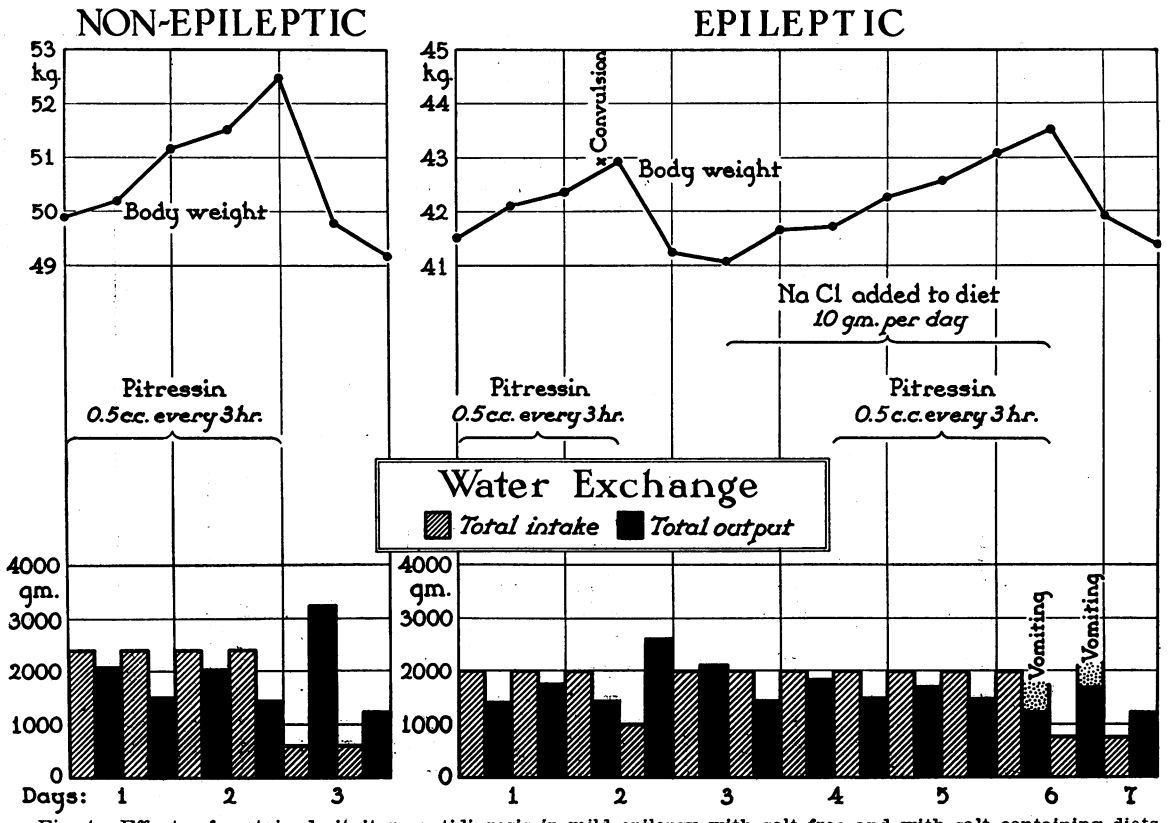

Fig. 4.—Effects of sustained pituitary antidiuresis in mild epilepsy with salt-free and with salt-containing diets. (See text.)

narcosis. When given with a convulsant drug, such as strychnin, lecithin diminishes the convulsive dose. It likewise tends to cause a retention of water in the body. Several of the current theories of narcosis and sedation take into account equilibria involving the various cell lipids, particularly in connection with the concept of cell membrane permeability. Comparative analyses of the brain tissue of epileptic and non-epileptic subjects of similar ages are being carried out in our laboratory with the hope that they may shed additional light on this phase of our problem. In this connection it may be significant that brain tissue normally contains more than twice the concentration of cholesterol during adult life that it contains during early childhood, the period when susceptibility to convulsions is highest. The concentration of lecithin, on the other hand, is almost as great in the brain of the young child as in that of the adult.

Water-Balance.—The significance of the water-balance in relationship to the occurrence of seizures in epilepsy has long been suspected from the fact that the brains of patients dying in *status epilepticus* show an increased water content. Even Hippocrates appreciated this relationship, for in his classical treatise on "The Sacred Disease" he states, "Whoever is acquainted with such a change (epilepsy) in men, and can render a man humid and dry, hot and cold by regimen, could also cure this disease." It has recently been found to be true that procedures which cause a negative water-balance in the body (such as restriction of the water intake, diuresis, catharsis, profuse sweating, starvation, use of a ketogenic diet and administration of acid-forming salts) tend to prevent seizures. On the other hand, retention of water under certain conditions favors the occurrence of convulsions.

The mechanism of this relationship is not well understood. Fay,[7] who first reported the clinical effects of water-restriction on cases showing encephalographic evidence of cortical atrophy, maintains that the effect of water retention is purely mechanical. This belief is based on his assumption that disease of the Pacchionian bodies is characteristic of epilepsy, and that fluid accumulates under increased pressure in the cortical subarachnoid spaces because its absorption by way of these structures is impaired. These claims have not been substantiated by other workers.[8,9,10] The accompanying charts (Figures 2 and 3) show data from studies on the water and mineral balances in relationship to the occurrence of seizures, which permit of quite a different interpretation.[11]

The purpose of the study reported in Figure 2 was to determine the effect of changes in water-balance upon the occurrence of convulsions when other factors than water restriction were employed to modify the water exchange. The experimental subject was a thirteen-year-old girl who was known to have from two to fifteen convulsions daily when not under intensive treatment. She was kept in the metabolism ward on a constant non-ketogenic diet. It is obvious, from the upper portion of the chart, that the essential factor

TABLE 4.—*Parallelism Between Effects of Various Factors on Permeability of Brain Cell Membranes and on Occurrence of Convulsions in Epilepsy*

Permeability		Physiological Factor	Convulsions	
Increased	Decreased		Induced	Prevented
....	X	Narcosis	....	X
....	X	Sedation	....	X
....	X	Acidosis	....	X
....	X	Dehydration	....	X
....	X	Ketosis	....	X
....	X	High bl. cholesterol / bl. lecithin	....	X
X	....	Excitation	X	....
X	....	Trauma	X	....
X	....	Alkalosis	X	....
X ?	....	"Superhydration"	X	....
X	....	Anoxemia	X	....
X	....	Low bl. cholesterol / bl. lecithin	X	....
X	....	Hypocalcemia	X	....
X ?	....	Hypoglycemia	X	....

in the reaction is the net, rather than the total, water exchange. While a strongly negative water-balance, produced in a variety of ways, resulted in a cessation of seizures, these always recurred within twelve to thirty hours following the re-establishment of a positive balance. Even those which occurred at the beginning, and toward the end of the NH_4NO_3 period, actually followed temporary water storage, which is not indicated in the chart.

A simultaneous study of the mineral exchanges showed that sodium and chlorin were in negative balance during all periods, showing a net loss of body water, but were stored during periods of positive water-balance. Potassium excretion, on the other hand, was greatly increased during periods of water storage, a negative balance appearing even before the onset of seizures. This is illustrated in Figure 3, which shows the K, Na, Cl and water-balances just before, during and after the pitressin period. Since most of the potassium of the body occurs within the cells, it is practically certain that a strongly negative balance of this element indicated leakage from the cells into the extracellular fluids from which it is excreted, chiefly by way of the kidneys. A non-epileptic subject did not show this phenomenon under identical conditions. From these and other data, yet to be presented, it may be assumed that sudden reëstablishment of a positive water-balance, following a period of net loss, is accompanied in the epileptic subject by a severe disturbance of the electrolyte equilibria on the two sides of the brain cell membranes. The cells probably imbibe extra water under these conditions. The abnormal reaction is apparently accentuated by sustained pituitary antidiuresis.

In a special study of the latter effect in mildly epileptic, as contrasted with non-epileptic subjects, it was found that seizures could be induced almost at will in the majority of the former, but not at all in the latter by administration of posterior pituitary extract at three-hour intervals during periods of high water and low mineral intake.[12] That the seizures so induced were due to alterations in the water and electrolyte exchanges, rather than to the vasopressor action of the extract, was indicated by the fact that they occurred only after the amount of water forcibly retained became equal to between 2 and 5 per cent of the body weight. It has been shown that the extracellular body fluids undergo a considerable degree of dilution under these conditions. Prevention of this dilution by the simultaneous administration of just sufficient NaCl to bring the fluid retained to isotonic concentration interferes with the induction of seizures by this procedure, as illustrated in Figure 4. Prolonged dilution of the body fluids undoubtedly results in a disturbance of the electrolyte equilibria in a direction favorable to an increase in nervous irritability. The mechanism of the well-known therapeutic effect of bromids in epilepsy may ultimately find an explanation in the action of the Br ion in preventing dilution of the intracellular fluids of the brain. This is suggested by the recent demonstration by Notkin, Garcia and Killian,[13] that administration of bromids causes an increase of from 15 to 36 per cent in the total halogen concentration in blood cells. Preliminary studies made in our laboratory indicate that there may be a similar storage of Br in brain tissue, following intensive bromid medication.

The apparent migration of potassium ions from the body cells (brain) of the epileptic prior to and during the course of seizures, and the observation that the epileptic cannot withstand dilution of his extracellular body fluids without having

seizures, have together been interpreted as indirect evidence of defective brain cell membranes. Confirmatory of this tentative view is the fact that the majority of factors which tend to induce seizures are alleged to increase cell membrane permeability, while those which tend to prevent their occurrence are known to decrease permeability. These factors and their effects are summarized in Table 4.

When one surveys the conceivable defects of the brain cells which might reasonably account for the abnormal convulsive reactability of the epileptic patient, there is no other which appears to satisfy all of the requirements so well as that implied in the foregoing interpretation. As a working hypothesis, therefore, the generalization, that the epileptic differs from the normal subject in exhibiting an innate defect in his brain cell membranes, possesses some practical value in that all therapy may be directed toward correcting such defect or producing a decrease in permeability. It is obviously necessary to consider all of the foregoing etiologic factors in relationship to each other before accurate interpretation of a response to any single factor can be made. Under ordinary conditions there is present at any given time a multiplicity of contributing factors, some tending to provoke, others to prevent seizures. Whether or not a seizure will occur, then, depends upon the relative strength of the provocative as against the preventive forces.

From the meager data already available regarding the brain cell changes in the non-organic convulsive disorders other than cryptogenic epilepsy, it can merely be remarked that there apparently tends to be an increased water content in relationship to seizures in those types which have been studied. Drabkin [14] has shown that the brain swells due to water imbibition before convulsions occur from insulin overdosage. Preliminary dehydration prevents such convulsions. Baar [15] observed an increased water content in the brain tissue of subjects with infantile tetany, and considers this increased hydration to be of primary significance in the causation of convulsions in this condition. Ellis [16] independently reported a similar increase in the water content of the brain in guanadine tetany and in experimental hypoparathyroid tetany. We have recently determined that a positive water-balance favors the occurrence of convulsions in a case of idiopathic parathyroid tetany. The "hard" edema (intracellular?) of the brain found in cases of acute nephritic uremia and in eclampsia of pregnancy, is probably of considerable importance in the convulsive mechanism in these disorders. Alkalosis tends to provoke convulsions in latent infantile or parathyroid tetany, while acidosis has the reverse effect. The effect of calcium deficiency in the body fluids in tetany may be responsible for a decrease in nerve cell membrane permeability; but, if this exists at all, it must be temporary, disappearing in response to antitetany therapy.

(To be continued)

HOW CAN PSYCHIATRY PROGRESS?*

By CLIFFORD W. MACK, M. D.
Livermore

THE honor of being chairman of this section carries a penalty, or rather an obligation, to present something at the meeting that furthers progress in our specialty. The chairman is allowed by custom to cover a wide range, rather than choose the limitation of a definite scientific subject. If, by taking advantage of this privilege, the remarks are discursive, it is for the purpose of pointing out some of the pressing needs confronting us.

THE NEUROPSYCHIATRIC SECTION

My responsibility would not be adequately fulfilled if I did not say something pertaining to the welfare, growth and development of this section of the California State Medical Society. We all have a duty that should not be left entirely to the officers who come and go each year, to make this section an important scientific body of the parent society. The importance of neuropsychiatry in the practice of medicine is well recognized and needs no defense. The men and women in the state engaged in this branch of medicine should take a greater personal interest in our meetings by attendance and participation in the discussions. The papers presented should only be the introduction that develops a free interchange of ideas. The members of the state and government hospital staffs are earnestly urged to take a more active part in the affairs of this section, as their presence at the meetings would add to the value of the scientific program, with benefit to themselves and their associates in the field of psychiatry. These members of the section have under their care in the state hospitals half of all the hospital patients in the state. The medical profession is more and more looking to psychiatry for assistance in clinical work. The public social agencies, and the various educators are calling upon psychiatry to lead in the solution of some of the problems of human behavior. In addition to all of this, there are a large number of patients going through the private offices of neuropsychiatrists as mild psychoses and psychoneuroses. The sum total of our endeavors is a vast part in the care of sick and handicapped people, and hence our section should be one of the most important in the society.

The section was organized on May 20, 1916. It is worthy of mention that the number engaged in our specialty in California, as judged by membership in the American Psychiatric Association, has increased from nineteen in 1913 to fifty-nine in 1933. The work of the section should proportionately increase so that it will be truly representative of the large field which it covers.

STATE HOSPITAL SERVICE

Mindful of my days in state hospital service, I cannot neglect the duty of calling your attention to the obligation we have toward those hundreds

* Chairman's address, Neuropsychiatry Section of the California Medical Association, at the sixty-third annual session, Riverside, April 30 to May 3, 1934.